10676089

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(FILE 'HOME' ENTERED AT 13:56:05 ON 07 JUL 2005)

FILE 'MEDLINE' ENTERED AT 13:56:13 ON 07 JUL 2005

L1	11937	S	RETINOID?
L2	2642	S	RAR
L3	13200	S	L1 OR L2
L4	4876	S	ATOPY
L5	4	S	L3 AND L4
L6	48923	S	DERMATITIS
L7	0	S	L6 AND LL3
L8	117	S	L6 AND L3
L9	23	S	ECZEMA AND L3
L10	629	S	L3 AND PSORIASIS
L11	422	S	L3 AND ACNE
		E	BIOSCI

FILE 'EMBASE' ENTERED AT 14:05:09 ON 07 JUL 2005

L12	14	S	(RETINOID OR RAR) AND ATOPY

10676089

ANSWER 1 OF 4 MEDLINE on STN

AN 2003044756 MEDLINE

DN PubMed ID: 12553849

TI Ichthyosis: etiology, diagnosis, and management.

AU DiGiovanna John J; Robinson-Bostom Leslie

CS Division of Dermatopharmacology, Brown Medical School and Rhode Island Hospital, Providence 02903, USA.. John_DiGiovanna_MD@Brown.edu

SO American journal of clinical dermatology, (2003) 4 (2) 81-95. Ref: 100
Journal code: 100895290. ISSN: 1175-0561.

CY New Zealand

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 200306

ED Entered STN: 20030130
Last Updated on STN: 20030620
Entered Medline: 20030619

AB The ichthyoses are a heterogeneous group of disorders with both inherited and acquired forms. Clinical presentation, pattern of inheritance, and laboratory evaluation may establish a precise diagnosis, which can assist in prognosis and genetic counseling. Congenital autosomal recessive ichthyosis (CARI) usually presents at birth, often as a collodion baby. CARI can progress into any one of a spectrum of disorders. Lamellar ichthyosis is characterized by dark, plate (armor)-like scale. This disease is often caused by mutations in the gene encoding the enzyme transglutaminase 1. Congenital ichthyosiform erythroderma is another phenotype within CARI, marked by generalized redness and fine white scale. Epidermolytic hyperkeratosis is an autosomal dominant disorder characterized by hyperkeratosis and blistering, and at least six clinical phenotypes have been described. It may be due to mutations in the gene encoding the intermediate filament proteins keratin 1 and 10. Ichthyosis vulgaris is the most common ichthyosis, and is inherited in an autosomal dominant pattern. Involvement is generally mild and may vary greatly with climate and humidity. X-linked ichthyosis, due to a defect in the enzyme steroid sulfatase, affects males with generalized scaling that usually begins soon after birth. There may be associated corneal opacities that do not affect vision. Sjogren-Larsson syndrome is an autosomal recessive ichthyosis associated with progressive spastic paralysis and mental retardation. This condition is caused by mutations in the gene for fatty aldehyde dehydrogenase. Refsum's disease, due to accumulation of phytanic acid, results in ichthyosis and progressive neurologic dysfunction. The erythrokeratodermas are characterized by hyperkeratosis and localized erythema. Erythrokeratodermia variabilis is autosomal dominant and characterized by generalized or localized hyperkeratosis and migratory red patches. Mutations in the genes encoding the gap junction proteins, connexins, underlie this disorder. Netherton's syndrome is an autosomal recessive disorder characterized by ichthyosis, a hair shaft abnormality and **atopy.** The ichthyosis may present at birth with erythroderma or in some cases a collodion presentation. However, a frequent characteristic skin manifestation is ichthyosis linearis circumflexa. Netherton's syndrome has been found to be due to an abnormality in a serum protease inhibitor. Acquired ichthyosis can have a variety of underlying causes including neoplastic, infectious, drugs, endocrine, metabolic, autoimmune, malabsorptive states, and hereditary. Topical, and in more severe cases, systemic, therapy are useful in managing this array of disorders of cornification.

AB . . . proteins, connexins, underlie this disorder. Netherton's syndrome is an autosomal recessive disorder characterized by ichthyosis, a hair shaft abnormality and **atopy.** The ichthyosis may present at birth with erythroderma or in some cases a collodion presentation. However, a frequent characteristic skin. . .

CT . . . ET, etiology
*Ichthyosis: TH, therapy
Keratolytic Agents: TU, therapeutic use
Lubrication
Medical History Taking
Physical Examination
Research Support, Non-U.S. Gov't
Retinoids: TU, therapeutic use

CN 0 (Keratolytic Agents); 0 (**Retinoids**)

L5 ANSWER 2 OF 4 MEDLINE on STN
AN 2001455618 MEDLINE
DN PubMed ID: 11502488
TI Could bronchial asthma be an endogenous, pulmonary expression of **retinoid** intoxication?.
AU Mawson A R
CS College of Health Sciences, Des Moines University-Osteopathic Medical Center, 3200 Grand Avenue, Des Moines, Iowa 50312, USA.. anthony.mawson@dmu.edu
SO Frontiers in bioscience : a journal and virtual library, (2001 Aug 1) 6 D973-85. Electronic Publication: 2001-08-01. Ref: 124
Journal code: 9709506. ISSN: 1093-4715.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20010815
Last Updated on STN: 20020122
Entered Medline: 20011213

AB Asthma has become a major public health problem, affecting about 17 million people in the United States, including 4.8 million children. A striking increase in asthma and other forms of **atopy** has occurred in children in the U.S. and other western countries during the past 30 years. Several studies have reported an inverse association between childhood infectious illness and the development of **atopy** , suggesting that certain forms of infection protect against and even inhibit asthma. This may involve a shift in the balance of CD4 T lymphocyte helper cells from a Th2 to a Th1-type cytokine profile. However, the underlying mechanisms remain uncertain. Based on a review of the literature, it is conjectured that in the absence of certain types of childhood infection, **retinoids** (vitamin A and its congeners) accumulate in the lung. Later, upon exposure to known triggers for asthma, **retinoid** metabolites may be produced in such high concentration that they produce an acute, localized form of **retinoid** intoxication, recognized as status asthmaticus.

TI Could bronchial asthma be an endogenous, pulmonary expression of **retinoid** intoxication?.

AB . . . 17 million people in the United States, including 4.8 million children. A striking increase in asthma and other forms of **atopy** has occurred in children in the U.S. and other western countries during

the past 30 years. Several studies have reported an inverse association between childhood infectious illness and the development of **atopy**, suggesting that certain forms of infection protect against and even inhibit asthma. This may involve a shift in the balance. . . Based on a review of the literature, it is conjectured that in the absence of certain types of childhood infection, **retinoids** (vitamin A and its congeners) accumulate in the lung. Later, upon exposure to known triggers for asthma, **retinoid** metabolites may be produced in such high concentration that they produce an acute, localized form of **retinoid** intoxication, recognized as status asthmaticus.

L5 ANSWER 3 OF 4 MEDLINE on STN
AN 89340009 MEDLINE
DN PubMed ID: 2527214
TI [Successful **retinoid** therapy of Netherton syndrome].
Erfolgreiche **Retinoidtherapie** des Netherton-Syndroms.
AU Hartschuh W; Hausser I; Petzoldt D
CS Hautklinik, Ruprecht-Karls-Universitat Heidelberg.
SO Der Hautarzt; Zeitschrift fur Dermatologie, Venerologie, und verwandte Gebiete, (1989 Jul) 40 (7) 430-3.
Journal code: 0372755. ISSN: 0017-8470.
CY GERMANY, WEST: Germany, Federal Republic of
DT (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 198909
ED Entered STN: 19900309
Last Updated on STN: 20020125
Entered Medline: 19890919
AB A young patient with Netherton's syndrome characterized by the classic triad of ichthyosis linearis circumflexa, trichorhexis invaginata and **atopy** was treated with Acitretin, a new **retinoid** preparation: 35 mg Acitretin/day resulted in a severe, erosive dermatitis which necessitated interruption of therapy. Even with 10 mg/day the patient had intolerable irritation of the integument. After a further dosage reduction to 5 mg/day there were no obvious side effects and a long-term treatment was possible, resulting in an obvious reduction of the ichthyotic lesions and improved hair growth. Electron microscopy in the active part of the skin lesions from untreated skin revealed granular, membrane-enclosed material intracellularly and in the intercellular spaces of the granular layer. Keratinization was almost completely suppressed. Therapy with Acitretin drastically reduced the deposition of intra- and extracellular material and normalized keratinization. Our results underline the importance of starting **retinoid** therapy in Netherton's syndrome at a low dosage and adjusting it carefully in each case with reference to the skin manifestations and the side effects.
TI [Successful **retinoid** therapy of Netherton syndrome].
Erfolgreiche **Retinoidtherapie** des Netherton-Syndroms.
AB A young patient with Netherton's syndrome characterized by the classic triad of ichthyosis linearis circumflexa, trichorhexis invaginata and **atopy** was treated with Acitretin, a new **retinoid** preparation: 35 mg Acitretin/day resulted in a severe, erosive dermatitis which necessitated interruption of therapy. Even with 10 mg/day the. . . Acitretin drastically reduced the deposition of intra- and extracellular material and normalized keratinization. Our results underline the importance of starting **retinoid** therapy in Netherton's syndrome at a low dosage and adjusting it carefully in each

case with reference to the skin. . .

L5 ANSWER 4 OF 4 MEDLINE on STN
AN 84192681 MEDLINE
DN PubMed ID: 6718040
TI [The Netherton syndrome: clinical characteristics, differential diagnosis and new ways of therapy].
Das Netherton-Syndrom: Klinische Charakteristik, differential-diagnostische Abgrenzung und neue Wege der Therapie.
AU Haas O A; Martins da Cunha A; Gadner H; Stingl G; Kornmuller R
SO Padiatrie und Padologie, (1984) 19 (2) 153-9.
Journal code: 0022370. ISSN: 0030-9338.
CY Austria
DT (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 198406
ED Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19840613
AB The Netherton-syndrome is a rare disease which is probably inherited through an autosomal recessive trait. It is defined by a triad of symptoms: congenital ichthyosiform erythrodermia , trichorrhexis invaginata et nodosa ("bamboo hair") and **atopy.** Additional disorders affect the immune system, the metabolism of amino acids and the physical development. On the basis of a new case, the cellular immune defect and the genetic background of the disease are more clearly defined. A new form of treatment--a combination of photochemotherapy (PUVA) and systematic application of aromatic **retinoid**--has so far proved to be successful. In order to establish an accurate diagnosis--a prerequisite for this promising therapeutic approach--diseases with similar symptoms are discussed for comparison.
AB . . . trait. It is defined by a triad of symptoms: congenital ichthyosiform erythrodermia , trichorrhexis invaginata et nodosa ("bamboo hair") and **atopy.** Additional disorders affect the immune system, the metabolism of amino acids and the physical development. On the basis of a. . . the disease are more clearly defined. A new form of treatment--a combination of photochemotherapy (PUVA) and systematic application of aromatic **retinoid**--has so far proved to be successful. In order to establish an accurate diagnosis--a prerequisite for this promising therapeutic approach--diseases with. . .

10676089

9 ANSWER 5 OF 23 MEDLINE on STN
AN 2004033620 MEDLINE
DN PubMed ID: 14733065
TI [Topical immunomodulators for treatment of **eczema**].
Topische Immunmodulatoren zur Behandlung von Ekzemen.
AU Heine Guido; Sterry Wolfram; Worm Margitta
CS Klinik fur Dermatologie, Venerologie und Allergologie, Campus Charite Mitte, Universitatsklinikum der Humboldt-Universitat zu Berlin, Deutschland.
SO Wiener medizinische Wochenschrift (1946), (2003) 153 (23-24) 522-5. Ref: 24
Journal code: 8708475. ISSN: 0043-5341.
CY Austria
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA German
FS Priority Journals
EM 200406
ED Entered STN: 20040122
Last Updated on STN: 20040626
Entered Medline: 20040625
AB Anti-inflammatory treatment of eczematous skin diseases like atopic dermatitis and allergic contact dermatitis has mainly been performed with topical glucocorticosteroids. Increasing knowledge of the pathophysiological interactions and the immunological mechanisms during the chronic inflammatory processes in the skin offers new therapeutical options. In this review, new therapeutical approaches for the treatment of eczematous skin disease will be presented. These novel compounds include the topical immunomodulators, tacrolimus and pimecrolimus. Such molecules inhibit intracellular signal transducing phosphatases and act consecutively at the molecular level by inhibiting the activation of transcription factors. Secondly, the development of nuclear hormone receptor family members, such as **retinoids**, vitamin D and peroxisome proliferator-activated receptor agonists, is discussed. Substances from this family have differentiating, antiproliferative, but also immuno-modulatory effects, which make them attractive as anti-eczematous therapeutic compounds. The diversity of these interactions is extensive, and clinical studies will prove their clinical efficacy.
TI [Topical immunomodulators for treatment of **eczema**].
Topische Immunmodulatoren zur Behandlung von Ekzemen.
AB . . . molecular level by inhibiting the activation of transcription factors. Secondly, the development of nuclear hormone receptor family members, such as **retinoids**, vitamin D and peroxisome proliferator-activated receptor agonists, is discussed. Substances from this family have differentiating, antiproliferative, but also immuno-modulatory effects,. . .
CT *Adjuvants, Immunologic: AD, administration & dosage
Administration, Topical
*Dermatitis, Atopic: DT, drug therapy
Dermatitis, Atopic: IM, immunology
Drug Therapy, Combination
***Eczema: DT, drug therapy**
Eczema: IM, immunology
English Abstract
Humans

L9 ANSWER 6 OF 23 MEDLINE on STN
AN 2003519881 MEDLINE
DN PubMed ID: 14597015
TI Common pediatric and adolescent skin conditions.
AU Sanfilippo Angela M; Barrio Victoria; Kulp-Shorten Carol; Callen Jeffrey P
CS University of Louisville School of Medicine, Louisville, Kentucky 40202, USA.
SO Journal of pediatric and adolescent gynecology, (2003 Oct) 16 (5) 269-83. Ref: 39
Journal code: 9610774. ISSN: 1083-3188.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200405
ED Entered STN: 20031105
Last Updated on STN: 20040511
Entered Medline: 20040510
AB Skin lesions are encountered in all areas of medicine, and it is therefore important for physicians to understand the fundamentals of explaining and diagnosing common skin conditions. This article begins with a discussion of description and documentation of skin lesions based on color, size, morphology, and distribution. Pigmentation disorders such as vitiligo are depicted.Cutaneous growths that are found in the pediatric and adolescent population include acrochordons, dermatofibromas, keloids, milia, neurofibromas, and pyogenic granulomas. Treatment of these growths usually involves observation or curettage with electrodessication.Psoriasis, atopic dermatitis, poison ivy, and **eczema** are comprised of scaling patches and plaques; poison ivy and atopic dermatitis may also present with bullous and vesicular changes. Therapy typically consists of topical emollients and corticosteroids; phototherapy is reserved for refractory cases.Acne vulgaris is the most common skin disease of the pediatric and adolescent population. This condition can be psychologically debilitating and, therefore, proper treatment is of paramount importance. Therapeutic options include topical as well as oral antibiotics and **retinoids.** Extreme caution must be used when prescribing **retinoids** to post-pubescent females, as these agents are teratogenic.Vascular anomalies are most commonly exemplified as port wine stains and hemangiomas. Port wine stains may be treated with pulsed dye laser or may be observed if they are not of concern to the patient or physician. Hemangiomas typically spontaneously regress by age ten; however, there has been recent concern that certain cases may need to be treated.Dermal rashes may be localized or generalized. Treatment of generalized drug eruptions involves elimination of the inciting agent, topical antipruritics, and systemic corticosteroids for severe reactions.Infectious etiologic agents of skin disease include bacteria, fungi, and viruses. Many sexually transmitted diseases are bacterial or viral in origin and present as a rash or ulcer. Impetigo is a bacterial infection which may present as a bullous eruption or as an erosion with a honey colored crust. Other bacterial infections include erythema chronicum migrans, folliculitis, and cellulitis. Fungal infections include the various forms of tinea and are usually treated with topical antifungals; if the infection is located in a hair-bearing area, systemic antifungals are necessary. Viral infections include warts, varicella, molluscum contagiosum, and herpes. Treatment varies from observation or antivirals for varicella to cryosurgery and topical

imiquimod for warts. Finally, scabies and lice are infectious agents that can be treated with permethrin and pyrethrin solutions.

AB . . . neurofibromas, and pyogenic granulomas. Treatment of these growths usually involves observation or curettage with electrodessication.Psoriasis, atopic dermatitis, poison ivy, and **eczema** are comprised of scaling patches and plaques; poison ivy and atopic dermatitis may also present with bullous and vesicular changes.. . . psychologically debilitating and, therefore, proper treatment is of paramount importance. Therapeutic options include topical as well as oral antibiotics and **retinoids.** Extreme caution must be used when prescribing **retinoids** to post-pubescent females, as these agents are teratogenic.Vascular anomalies are most commonly exemplified as port wine stains and hemangiomas. Port. . .

L9 ANSWER 7 OF 23 MEDLINE on STN
AN 2002714612 MEDLINE
DN PubMed ID: 12476959
TI Novel therapies for atopic **eczema.**
AU Worm Margitta
CS Universitatsklinikum Charite Klinik for Dermatologie, Venerologie und Allergologie, Schumannstr 20-21, 10117 Berlin, Germany.. margitta.worm@charite.de
SO Current opinion in investigational drugs (London, England : 2000), (2002 Nov) 3 (11) 1596-603. Ref: 99
Journal code: 100965718. ISSN: 1472-4472.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200303
ED Entered STN: 20021217
Last Updated on STN: 20030327
Entered Medline: 20030326
AB Atopic dermatitis (AD) has been treated with topical glucocorticosteroids for decades. With the introduction of the topical immune modulators tacrolimus and pimecrolimus, a new treatment era has begun. The knowledge on pathophysiological interactions and immunological disturbances during the chronic inflammatory process in the skin has been continuously increasing and offers new therapeutical approaches. These are discussed in this review based on the current literature, my own research findings and recent patents. Development of members of the glucocortioid family such as **retinoids**, vitamin D and peroxisome proliferator-activated receptor agonists, are discussed. Molecules from members of this family have profound differentiating, antiproliferative, but also immunomodulatory effects, which make them attractive as anti-eczematous compounds. Furthermore, several anti-infective and antipruritic agents, and preparations which enhance thedisturbed skin barrier function in AD are presented. Phytopharmacological and miscellaneous approaches, including Chinese tea or gamma-linolenic acid, will be critically discussed. Finally, recently patented, experimental compounds are presented, which interfere with several pathways involved in the immune response of AD.
TI Novel therapies for atopic **eczema.**
AB . . . on the current literature, my own research findings and recent patents. Development of members of the glucocortioid family such as **retinoids**, vitamin D and peroxisome proliferator-activated

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12 ANSWER 1 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2005087725 EMBASE
TI A photo quiz to hone dermatologic skills.
AU Kaplan D.L.
CS Dr. D.L. Kaplan, University of Missouri, Kansas City, MO, United States
SO Consultant, (2004) Vol. 44, No. 9, pp. 1209-1216.
ISSN: 0010-7069 CODEN: CNSLAY
CY United States
DT Journal; Article
FS 013 Dermatology and Venereology
037 Drug Literature Index
LA English
ED Entered STN: 20050310
Last Updated on STN: 20050310
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER
CT Medical Descriptors:
*molluscum . . . disease: DI, diagnosis
clinical feature
anamnesis
rosacea
skin biopsy
sun exposure
tinea corporis
lupus erythematosus
hair loss
anemia
differential diagnosis
ovary polycystic disease
stress
Staphylococcus aureus
Staphylococcus infection: DT, drug therapy
Staphylococcus infection: ET, etiology
verruca vulgaris
atopy
papule: DT, drug therapy
cryotherapy
curettage
human
female
case report
human tissue
preschool child
adult
article
priority journal
antibiotic agent: DT, drug therapy
antibiotic agent: TP, topical drug administration
antifungal agent: DT, drug therapy
antifungal agent: TP, topical drug administration
retinoid
pseudomonic acid: DT, drug therapy
pseudomonic acid: TP, topical drug administration
retinoic acid: DT, drug therapy
retinoic acid: TP, topical drug administration
cimetidine: DT, drug. . .

L12 ANSWER 2 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.

on STN
AN 2004469839 EMBASE
TI [Topical treatment of dry and hyperkeratotic skins].
TRAITEMENT TOPIQUE DES PEAUX SECHES ET HYPERERATOSIQUES.
AU Gassia V.; Herve N.
CS V. Gassia, Dermatologue, 23, allees Charles de Fitte, 31300 Toulouse, France
SO Nouvelles Dermatologiques, (2004) Vol. 23, No. 8 SPEC. ISS., pp. 1-19.
Refs: 12
ISSN: 0752-5370 CODEN: NODEE2
CY France
DT Journal; General Review
FS 013 Dermatology and Venereology
037 Drug Literature Index
038 Adverse Reactions Titles
LA French; English
SL English; French
ED Entered STN: 20041129
Last Updated on STN: 20041129
AB The cutaneous physiology of dry and hyperkeratotic skins is reviewed when examining the role of the different layers of the skin. The systems permitting regulation of hydration are described. The active substances used to hydrate the skin are described, particularly humectants (glycerol, sorbitol, propylene glycol). urea, which is a physiological constituent of the stratum corneum, film-producing surfactants and the epidermal lipid counter-types. Dry skin is studied by disease: cutaneous xeroses, ichtyoses, atopic dermatites, psoriasis and hyperkeratoses. Treatment methods used to combat skin dryness are described. To illustrate this scientific part, answers to questions often asked by patients are presented.
CT Medical Descriptors:
*dry . . . filament
skin permeability
desquamation
cell differentiation
lipid membrane
diffusion
thermoregulation
epidermis
xerosis: SI, side effect
xerosis: TH, therapy
ichthyosis: DT, drug therapy
ichthyosis: TH, therapy
nephrotoxicity: SI, side effect
metabolic acidosis: SI, side effect
atopy
atopic dermatitis: ET, etiology
atopic dermatitis: TH, therapy
psoriasis: DT, drug therapy
psoriasis: TH, therapy
drug safety
drug efficacy
human
review
*glycerol
*sorbitol
*propylene glycol: DT, drug therapy
*propylene glycol: TP,. . . endogenous compound

water: EC, endogenous compound
hyaluronic acid: EC, endogenous compound
citrulline: EC, endogenous compound
petrolatum
pyroglutamic acid: EC, endogenous compound
hypocholesterolemic agent: AE, adverse drug reaction
retinoid: AE, adverse drug reaction
clofazimine: AE, adverse drug reaction
lithium carbonate: AE, adverse drug reaction
allopurinol: AE, adverse drug reaction
hydroxyurea: AE, adverse drug. . .

L12 ANSWER 3 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2004456349 EMBASE
TI Modulation of cytokine production by low and high **retinoid** diets in ovalbumin-sensitized mice.
AU Ruhl R.; Garcia A.; Schweigert F.J.; Worm M.
CS R. Ruhl, Institute of Nutritional Science, University of Potsdam, Potsdam-Rehbrucke, Germany
SO International Journal for Vitamin and Nutrition Research, (2004) Vol. 74, No. 4, pp. 279-284.
Refs: 24
ISSN: 0300-9831 CODEN: IJVNAP
CY Switzerland
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20041112
Last Updated on STN: 20041112
AB Retinoids modulate many physiological processes such as the differentiation and growth of different cell types, including cells from the immune system. We have previously shown that retinoids modulate IgE production in vitro and in vivo. In the present study we investigated the effects of retinoids in non-sensitized and ovalbumin-sensitized mice that were fed for 11 weeks with three different vitamin A (VA) diets: a) VA-deficiency diet, b) base diet, and c) base diet supplemented with 0.5% all-trans-retinoic acid (ATRA). Phorbol-myristate-acetate (PMA)/ionomycin-stimulated SMC (splenic mononuclear cells) from mice fed with ATRA and the vitamin A-deficient diet group showed increased interleukin-4 (IL-4) responses in non-sensitized mice. After ovalbumin sensitization in the VA-deficient and the ATRA supplementation diet groups, no significant effects on IL-4 production were observed. By contrast, gamma interferon (IFN-γ production from PMA/ionomycin-stimulated SMC was enhanced in the VA-deficient diet group in ovalbumin-sensitized mice, and also in non-sensitized mice compared to the base and the ATRA-supplemented diet group. The data indicate that VA and **retinoid** content in a diet influences the cytokine response in non-sensitized and also ovalbumin-sensitized mice. Therefore these molecules may serve as active modulators of cytokine production in vivo that are responsible for the induction and persistence of atopic diseases.
TI Modulation of cytokine production by low and high **retinoid** diets in ovalbumin-sensitized mice.
AB . . . and also in non-sensitized mice compared to the base and the ATRA-supplemented diet group. The data indicate that VA and **retinoid** content in a diet influences the cytokine response in

non-sensitized and also ovalbumin-sensitized mice. Therefore these molecules may serve as. . .

CT Medical Descriptors:
*cytokine production
*vitamin intake
*dietary intake
cell differentiation
cell growth
cell type
immune system
antibody production
sensitization
vitamin supplementation
retinol deficiency
in vivo study
atopy
in vitro study
spleen cell
mononuclear cell
nonhuman
female
mouse
animal experiment
controlled study
animal cell
article
*cytokine: EC, endogenous compound
***retinoid**
*ovalbumin
gamma interferon: EC, endogenous compound
immunoglobulin E: EC, endogenous compound
interleukin 4: EC, endogenous compound
phorbol 13 acetate 12 myristate
ionomycin

L12 ANSWER 4 OF 14 EMBASE
on STN
AN 2003345339 EMBASE
TI Early development of multiple epithelial neoplasms in Netherton syndrome.
AU Krasagakis K.; Ioannidou D.J.; Stephanidou M.; Manios A.; Panayiotides J.G.; Tosca A.D.
CS Dr. K. Krasagakis, Department of Dermatology, Univ. General Hospital of Heraklion, GR-71110 Heraklion, Crete, Greece. krasagak@med.uoc.gr
SO Dermatology, (2003) Vol. 207, No. 2, pp. 182-184.
Refs: 18
ISSN: 1018-8665 CODEN: DERAEG
CY Switzerland
DT Journal; Article
FS 013 Dermatology and Venereology
016 Cancer
037 Drug Literature Index
LA English
SL English
ED Entered STN: 20030911
Last Updated on STN: 20030911
AB We report a case of Netherton syndrome manifested as congenital ichthyosiform erythroderma, trichorrhexis invaginata and **atopy**, who in early adulthood developed multiple, aggressive epithelial neoplasms

in sun-exposed areas of the skin, in areas with papillomatous skin hyperplasia and at the left parotid region. The occurrence of cutaneous neoplasia has been reported in syndromes with congenital ichthyosis and suggests that the underlying genetic defects may cause the development of cancer in prone patients. Copyright .COPYRGT. 2003 S. Karger AG, Basel.

AB We report a case of Netherton syndrome manifested as congenital ichthyosiform erythroderma, trichorrhexis invaginata and **atopy**, who in early adulthood developed multiple, aggressive epithelial neoplasms in sun-exposed areas of the skin, in areas with papillomatous skin. . .

CT Medical Descriptors:
*Netherton . . . DT, drug therapy
*multiple epithelial neoplasm: SU, surgery
*neoplasm: CO, complication
*neoplasm: DI, diagnosis
*neoplasm: DT, drug therapy
*neoplasm: SU, surgery
acute disease
disease course
clinical feature
congenital ichthyosiform erythroderma
trichorrhexis
atopy
sun exposure
skin
papillomatous skin hyperplasia
hyperplasia
parotid gland
incidence
skin tumor
drug megadose
disease activity
family history
histology
follow up
medical examination
laboratory test
diagnostic imaging
plastic surgery
human
male
case report
adult
article
priority journal
corticosteroid: DO, drug dose
corticosteroid: DT, drug therapy
retinoid: DT, drug therapy
antibiotic agent: DT, drug therapy
etretin: DT, drug therapy

L12 ANSWER 5 OF 14 EMBASE
on STN
AN 2003094283 EMBASE
TI Ichthyosis: Etiology, diagnosis, and management.
AU DiGiovanna J.J.; Robinson-Bostom L.
CS Dr. J.J. DiGiovanna, Department of Dermatology, Brown Medical School, Rhode Island Hospital, 593 Eddy Street, Providence, JBS-1, RI 02903, United States. John_DiGiovanna_MD@Brown.edu

10676089

SO American Journal of Clinical Dermatology, (2003) Vol. 4, No. 2, pp. 81-95.
Refs: 100
ISSN: 1175-0561 CODEN: AJCDCI
CY New Zealand
DT Journal; General Review
FS 013 Dermatology and Venereology
022 Human Genetics
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
ED Entered STN: 20030313
Last Updated on STN: 20030313
AB The ichthyoses are a heterogeneous group of disorders with both inherited and acquired forms. Clinical presentation, pattern of inheritance, and laboratory evaluation may establish a precise diagnosis, which can assist in prognosis and genetic counseling. Congenital autosomal recessive ichthyosis (CARI) usually presents at birth, often as a collodion baby. CARI can progress into any one of a spectrum of disorders. Lamellar ichthyosis is characterized by dark, plate (armor)-like scale. This disease is often caused by mutations in the gene encoding the enzyme transglutaminase 1. Congenital ichthyosiform erythroderma is another phenotype within CARI, marked by generalized redness and fine white scale. Epidermolytic hyperkeratosis is an autosomal dominant disorder characterized by hyperkeratosis and blistering, and at least six clinical phenotypes have been described. It may be due to mutations in the gene encoding the intermediate filament proteins keratin 1 and 10. Ichthyosis vulgaris is the most common ichthyosis, and is inherited in an autosomal dominant pattern. Involvement is generally mild and may vary greatly with climate and humidity. X-linked ichthyosis, due to a defect in the enzyme steroid sulfatase, affects males with generalized scaling that usually begins soon after birth. There may be associated corneal opacities that do not affect vision. Sjogren-Larsson syndrome is an autosomal recessive ichthyosis associated with progressive spastic paralysis and mental retardation. This condition is caused by mutations in the gene for fatty aldehyde dehydrogenase. Refsum's disease, due to accumulation of phytanic acid, results in ichthyosis and progressive neurologic dysfunction. The erythrokeratodermas are characterized by hyperkeratosis and localized erythema. Erythrokeratodermia variabilis is autosomal dominant and characterized by generalized or localized hyperkeratosis and migratory red patches. Mutations in the genes encoding the gap junction proteins, connexins, underlie this disorder. Netherton's syndrome is an autosomal recessive disorder characterized by ichthyosis, a hair shaft abnormality and **atopy.** The ichthyosis may present at birth with erythroderma or in some cases a collodion presentation. However, a frequent characteristic skin manifestation is ichthyosis linearis circumflexa. Netherton's syndrome has been found to be due to an abnormality in a serum protease inhibitor. Acquired ichthyosis can have a variety of underlying causes including neoplastic, infectious, drugs, endocrine, metabolic, autoimmune, malabsorptive states, and hereditary. Topical, and in more severe cases, systemic, therapy are useful in managing this array of disorders of cornification.
AB . . . proteins, connexins, underlie this disorder. Netherton's syndrome is an autosomal recessive disorder characterized by ichthyosis, a hair shaft abnormality and **atopy.** The ichthyosis may present at birth with erythroderma or in some cases a collodion presentation. However, a frequent characteristic skin. . .
CT Medical Descriptors:

*ichthyosis: . . . erythroderma: DT, drug therapy
ichthyosis vulgaris: DT, drug therapy
Sjoegren Larsson syndrome
autosomal dominant inheritance
autosomal recessive inheritance
climate
humidity
cornea opacity
spastic paresis
mental deficiency
Refsum disease
neurologic disease
erythema
Netherton disease
hair disease
atopy
skin manifestation
trichothiodystrophy
photosensitivity
family history
physical examination
laboratory diagnosis
hydration
lubrication
bath
drug efficacy
drug effect
skin irritation: SI, side effect
gene delivery system
drug elimination
calcification: SI, side effect
hyperostosis: SI, side effect
osteoporosis: SI, side effect
human
clinical trial
review
priority journal
***retinoid X receptor: EC, endogenous compound**
protein glutamine gamma glutamyltransferase: EC, endogenous compound
keratin: EC, endogenous compound
steryl sulfatase: EC, endogenous compound
aldehyde dehydrogenase: EC,. . . TP, topical drug administration
etretin: CM, drug comparison
etretin: DT, drug therapy
etretin: PK, pharmacokinetics
liarozole: DT, drug therapy
liarozole: PD, pharmacology
liarozole: PO, oral drug administration
retinoid: AE, adverse drug reaction
retinoid: CM, drug comparison
retinoid: DT, drug therapy
retinoid: PK, pharmacokinetics
retinoid: PD, pharmacology
unclassified drug

L12 ANSWER 6 OF 14 EMBASE
on STN
AN 2001339428 EMBASE

10676089

TI Enhanced lung C-fiber responsiveness in sensitized adult guinea pigs exposed to chronic tobacco smoke.

AU Bergren D.R.
CS D.R. Bergren, Dept. of Biomedical Sciences, School of Medicine, Creighton Univ., Omaha, NE 68178, United States. dbergren@creighton.edu

SO Journal of Applied Physiology, (2001) Vol. 91, No. 4, pp. 1645-1654.
Refs: 28
ISSN: 8750-7587 CODEN: JAPHEV
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 20011011
Last Updated on STN: 20011011

AB Tobacco smoke (TS) exposure induces bronchoconstriction and increases airway secretions and plasma extravasation in certain sensitive individuals, particularly those with asthma. C-fiber activation also induces these effects. Although the mechanism by which chronic TS exposure induces airway dysfunction is not well understood, TS exposure may enhance C-fiber responsiveness. To investigate the effect of chronic TS exposure on C-fiber responsiveness to capsaicin and bradykinin, especially in atopic individuals, we exposed ovalbumin (OA)-sensitized guinea pigs to TS (5 mg/l air, 30 min/day for 7 days/wk) or to compressed air. Nonsensitized guinea pigs were also exposed to either compressed air or TS. Beginning after 120 days of exposure, C fibers and rapidly adapting receptors (RARs) were challenged with capsaicin and bradykinin. TS exposure enhanced sensory receptor and airway responsiveness to both intravenous capsaicin and bradykinin challenge. C-fiber, **RAR**, and airway responsiveness to capsaicin challenge was greatest in OA-sensitized guinea pigs exposed to TS. OA alone induced capsaicin hyperresponsiveness at 5 μg. Airway responsiveness to bradykinin was also greatest in OA-sensitized guinea pigs exposed to TS. OA alone enhanced C-fiber responsiveness to bradykinin at 5 and 10 μg. C-fiber activation by either agonist appeared direct, whereas **RAR** activation appeared indirect. Therefore, a mechanism of airway hyperirritability induced by the combination of OA sensitization and chronic TS exposure may include hyperirritability of lung C fibers.

AB . . . with capsaicin and bradykinin. TS exposure enhanced sensory receptor and airway responsiveness to both intravenous capsaicin and bradykinin challenge. C-fiber, **RAR**, and airway responsiveness to capsaicin challenge was greatest in OA-sensitized guinea pigs exposed to TS. OA alone induced capsaicin hyperresponsiveness. . . OA alone enhanced C-fiber responsiveness to bradykinin at 5 and 10 μg. C-fiber activation by either agonist appeared direct, whereas **RAR** activation appeared indirect. Therefore, a mechanism of airway hyperirritability induced by the combination of OA sensitization and chronic TS exposure. . .

CT Medical Descriptors:
*nerve fiber C
guinea pig
bronchospasm
bronchus secretion
long term exposure
sensitization
sensory receptor
bronchus hyperreactivity
trachea pressure

atopy
agonist
nonhuman
male
animal experiment
controlled study
animal tissue
animal cell
article
priority journal
*tobacco smoke
capsaicin
bradykinin
ovalbumin

L12 ANSWER 7 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2000011967 EMBASE
TI [Netherton's-syndrome: An ichthyosiform dermatosis with hair abnormalities and atopic diathesis].
NETHERTON-SYNDROM: EINE ICHTHYOSIFORME GENODERMATOSE MIT HAARSCHAFTANOMALIE UND ATOPISCHER DIATHESE.
AU Sachs B.; Hertl M.
SO H+G Zeitschrift fur Hautkrankheiten, (1999) Vol. 74, No. 12, pp. 766-767.
Refs: 7
ISSN: 0301-0481 CODEN: ZHKRAJ
CY Germany
DT Journal; Conference Article
FS 007 Pediatrics and Pediatric Surgery
013 Dermatology and Venereology
022 Human Genetics
037 Drug Literature Index
LA German
SL English; German
ED Entered STN: 20000113
Last Updated on STN: 20000113
AB Netherton's syndrome is a rare disorder with an assumed autosomal recessive trait that consists of the triad trichorrhexis invaginata, ichthyosiform dermatosis and atopic diathesis. We report on a five years old female child with Netherton's syndrome, whose condition improved by **retinoid** therapy with acitretin at 0,5 mg/kg body weight.
AB . . . dermatosis and atopic diathesis. We report on a five years old female child with Netherton's syndrome, whose condition improved by **retinoid** therapy with acitretin at 0,5 mg/kg body weight.
CT Medical Descriptors:
*congenital ichthyosiform erythroderma: CN, congenital disorder
*congenital ichthyosiform erythroderma: DT, drug therapy
atopy: DT, drug therapy
syndrome delineation
autosomal recessive disorder
human
female
case report
child
conference paper
*etretin: DT, drug therapy
***retinoid: DT, drug therapy**

L12 ANSWER 8 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 97233254 EMBASE
DN 1997233254
TI Failure of cyclosporine in Netherton's syndrome [4].
AU Braun R.P.; Ramelet A.A.
CS Dr. A.A. Ramelet, 2 place Benjamin-Constant, CH-1003 Lausanne, Switzerland
SO Dermatology, (1997) Vol. 195, No. 1, pp. 75.
Refs: 19
ISSN: 1018-8665 CODEN: DERAEG
CY Switzerland
DT Journal; Letter
FS 013 Dermatology and Venereology
037 Drug Literature Index
LA English
ED Entered STN: 970822
Last Updated on STN: 970822
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER
CT Medical Descriptors:
*genodermatosis: DT, drug therapy
atopy: DT, drug therapy
case report
clinical feature
female
hair disease: DT, drug therapy
human
letter
oral drug administration
priority journal
puva
topical drug administration
treatment failure
*cyclosporin a: DT, drug therapy
etretin: DT, drug therapy
etretinate: DT, drug therapy
ibuprofen: DT, drug therapy
ketoconazole: DT, drug therapy
lactic acid: DT, drug therapy
metronidazole: DT, drug therapy
retinoid: DT, drug therapy
tetracosactide: DT, drug therapy
triamcinolone: DT, drug therapy

L12 ANSWER 9 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 96204897 EMBASE
DN 1996204897
TI Severe congenital generalized exfoliative erythroderma in newborns and infants: A possible sign of Netherton syndrome.
AU Hausser I.; Anton-Lamprecht I.
CS Department of Dermatology, Ultrastructure Res. Inst. of Skin, Voss-Str. 2,D-69115 Heidelberg, Germany
SO Pediatric Dermatology, (1996) Vol. 13, No. 3, pp. 183-199.
ISSN: 0736-8046 CODEN: PEDRDQ
CY United States
DT Journal; Article
FS 007 Pediatrics and Pediatric Surgery
013 Dermatology and Venereology

10676089

LA English
SL English
ED Entered STN: 960809
Last Updated on STN: 960809
AB We examined skin biopsy specimens from 17 of 19 newborns or infants with generalized ichthyosiform, exfoliative, seborrheic, or psoriasiform erythroderma. The specimens showed similar characteristic but nonspecific and therefore, at first sight, uninformative histologic features. Morphologically, the skin was affected overall with a persistent outbreak of eczema-like eruptions of subacute or chronic dermatitis. Pronounced dermal inflammatory processes were obvious by their perivascular and interstitial presence as well as exocytosis of lymphocytes, macrophages, and neutrophils. Epidermal barrier function was impaired by the highly suppressed terminal differentiation, with thin or in part completely absent stratum corneum, decrease of keratin filaments, decrease or lack of keratohyalin granules, and of keratinosomes containing stacks of lipid membranes. As a result, the formation and discharge of epidermal barrier lipids from the keratinosomes that normally provide intercellular lamellar sheets at the granular-horny layer interface contributing to the epidermal barrier, was highly disturbed. The concomitant loss of water, electrolytes, and proteins by fluid exudation caused the patients severe metabolic problems and recurrent infections. The suspicion of Netherton syndrome was eventually confirmed in 18 patients by light microscopic demonstration of bamboo hairs (trichorrhexis invaginata), mostly from the scalp, but also in vellus hairs and eyelashes. **Atopy** actually belongs to the symptom triad defining Netherton syndrome and is, in our opinion, primarily responsible for the pathologic events within the skin and of the keratinizing parts of the growing hair shafts. Differential expression of the atopic condition determines the appearance of the keratinization disorder of the skin, namely, severe, generalized, exfoliative erythroderma or milder forms of ichthyosis linearis circumflexa Comel. **Retinoid** treatment seems to be contraindicated in these conditions since their biopharmacologic effects involve suppression of terminal differentiation, which is the proper pathognomonic event. In six patients the condition had a fatal course within months because of hypernatremia, recurrent infections, failure to thrive, and sepsis. Our aim is to call attention to and reaffirm that in congenital or early infantile cases of generalized exfoliative erythroderma, Netherton syndrome should be suspected as the underlying disease.
AB . . . by light microscopic demonstration of bamboo hairs (trichorrhexis invaginata), mostly from the scalp, but also in vellus hairs and eyelashes. **Atopy** actually belongs to the symptom triad defining Netherton syndrome and is, in our opinion, primarily responsible for the pathologic events. . . . of the keratinization disorder of the skin, namely, severe, generalized, exfoliative erythroderma or milder forms of ichthyosis linearis circumflexa Comel. **Retinoid** treatment seems to be contraindicated in these conditions since their biopharmacologic effects involve suppression of terminal differentiation, which is the. . .
CT Medical Descriptors:
*congenital ichthyosiform erythroderma: CN, congenital disorder
*congenital ichthyosiform erythroderma: DI, diagnosis
article
atopy
clinical article
clinical examination
disease course

electron microscopy
female
human
infant
infant disease
male
newborn
newborn disease
priority journal
syndrome

L12 ANSWER 10 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 94344386 EMBASE
DN 1994344386
TI A clinical and immunological study of Netherton's syndrome.
AU Judge M.R.; Morgan G.; Harper J.I.
CS Department of Dermatology, The Hospitals for Sick Children, Great Ormond Street,London WC1N 3JH, United Kingdom
SO British Journal of Dermatology, (1994) Vol. 131, No. 5, pp. 615-621.
ISSN: 0007-0963 CODEN: BJDEAZ
CY United Kingdom
DT Journal; Article
FS 013 Dermatology and Venereology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
ED Entered STN: 941207
Last Updated on STN: 941207
AB Netherton's syndrome is a rare genodermatosis of unknown cause, which is classified as an ichthyosiform syndrome. A clinical and immunological study of seven patients with Netherton's syndrome illustrates the clinical spectrum of this disorder, the frequent association with **atopy**, and the absence of consistent immunological abnormalities. Failure to thrive in infancy was a feature in six of the seven patients, and was considered to be life-threatening in three. The skin disease evolved into ichthyosis linearis circumflexa in four of the seven, and the remaining three patients suffered from persistent or recurrent ichthyosiform erythroderma.
AB . . . and immunological study of seven patients with Netherton's syndrome illustrates the clinical spectrum of this disorder, the frequent association with **atopy**, and the absence of consistent immunological abnormalities. Failure to thrive in infancy was a feature in six of the seven. . .
CT Medical Descriptors:
*erythroderma: . . . drug administration
priority journal
topical drug administration
*emollient agent: DT, drug therapy
*etretin: DT, drug therapy
*immunoglobulin e: EC, endogenous compound
*immunoglobulin g: EC, endogenous compound
***retinoid: DT, drug therapy**
antihistaminic agent: DT, drug therapy
betamethasone valerate: DT, drug therapy
betamethasone valerate: AE, adverse drug reaction
corticosteroid: AE, adverse drug reaction

corticosteroid:. . .

L12 ANSWER 11 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 92090113 EMBASE
DN 1992090113
TI [**Atopy** and allergy: Scientific and therapeutic challenge of our time].
ATOPIE UND ALLERGIE: WISSENSCHAFTLICHE UND THERAPEUTISCHE HERAUSFORDERUNG UNSERER TAGE.
AU Borelli S.
CS Dermatologische Klinik und Poliklinik, Technische Universitat, Biedersteiner Str. 29, D-8000 Munchen 40, Germany
SO H+G Zeitschrift fur Hautkrankheiten, (1991) Vol. 66, No. SUPPL. 2, pp. 9-19.
ISSN: 0301-0481 CODEN: ZHKRAJ
CY Germany
DT Journal; Conference Article
FS 013 Dermatology and Venereology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA German
SL English; German
ED Entered STN: 920417
Last Updated on STN: 920417
AB We ask if, and if so why, atopic diseases may have grown in number during the last decades. Genetic and demographic factors do, in our opinion, outweigh the role of environmental pollution. We warn against setting high hopes in all to simple and thus dangerous therapies, e.g. rigid food-plans. Conversely we demonstrate the safe and long-term success of climatotherapy, as it can be achieved in the Deutsche Klinik fur Dermatologie und Allergie Davos - Alexanderhausklinik -. The high-mountain valley of Davos with it's combination of altitude radiation, low atmospheric humidity and it's light, but steady winds is the mainstay of successful therapy.
TI [**Atopy** and allergy: Scientific and therapeutic challenge of our time].
ATOPIE UND ALLERGIE: WISSENSCHAFTLICHE UND THERAPEUTISCHE HERAUSFORDERUNG UNSERER TAGE.
CT Medical Descriptors:
*asthma: . . .
therapy
fumaric acid: CB, drug combination
hydrocortisone acetate: DT, drug therapy
hydroxyzine: DT, drug therapy
loratadine: DT, drug therapy
methoxsalen: DT, drug therapy
promethazine: DT, drug therapy
retinoid: DT, drug therapy
tritoqualine: CB, drug combination
tritoqualine: DT, drug therapy
unclassified drug

L12 ANSWER 12 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 89175043 EMBASE
DN 1989175043

10676089

TI [Successful **retinoid** treatment of Netherton's syndrome].
ERFOLGREICHE RETINOIDTHERAPIE DES NETHERTON-SYNDROMS.
AU Hartschuh W.; Hausser I.; Petzoldt D.
CS Hautklinik, Ruprecht-Karls-Universitat, D-6900 Heidelberg, Germany
SO Hautarzt, (1989) Vol. 40, No. 7, pp. 430-433.
ISSN: 0017-8470 CODEN: HAUTAW
CY Germany
DT Journal
FS 013 Dermatology and Venereology
LA German
SL English
ED Entered STN: 911212
Last Updated on STN: 911212
AB A young patient with Netherton's syndrome characterized by the classic triad of ichthyosis linearis circumflexa, trichorhexis invaginata and **atopy** was treated with Acitretin, a new **retinoid** preparation: 35 mg Acitretin/day resulted in a severe, erosive dermatitis which necessitated interruption of therapy. Even with 10 mg/day the patient had intolerable irritation of the integument. After a further dosage reduction to 5 mg/day there were no ebvious side effects and a long-term treatment was possible, resulting in an obvious reduction of the ichthyotic lesions and improved hair growth. Electron microscopy in the active part of the skin lesions from untreated skin revealed granular, membrane-enclosed material intracellularly and in the intercellular spaces of the granular layer. Keratinization was almost completely suppressed. Therapy with Acitretin drastically reduced the deposition of intra- and extracellular material and normalized keratinization. Our results underline the importance of starting **retinoid** therapy in Netherton's syndrome at a low dosage and adjusting it carefully in each case with reference to the skin manifestations and the side effects.
TI [Successful **retinoid** treatment of Netherton's syndrome].
ERFOLGREICHE RETINOIDTHERAPIE DES NETHERTON-SYNDROMS.
AB A young patient with Netherton's syndrome characterized by the classic triad of ichthyosis linearis circumflexa, trichorhexis invaginata and **atopy** was treated with Acitretin, a new **retinoid** preparation: 35 mg Acitretin/day resulted in a severe, erosive dermatitis which necessitated interruption of therapy. Even with 10 mg/day the. . . Acitretin drastically reduced the deposition of intra- and extracellular material and normalized keratinization. Our results underline the importance of starting **retinoid** therapy in Netherton's syndrome at a low dosage and adjusting it carefully in each case with reference to the skin. . .

L12 ANSWER 13 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 84063802 EMBASE
DN 1984063802
TI Netherton's syndrome in two adult brothers.
AU Caputo R.; Vanotti P.; Bertani E.
CS First Clinic of Dermatology, University of Milan, 20122 Milan, Italy
SO Archives of Dermatology, (1984) Vol. 120, No. 2, pp. 220-222.
CODEN: ARDEAC
CY United States
DT Journal
FS 013 Dermatology and Venereology
022 Human Genetics
LA English
ED Entered STN: 911210

Last Updated on STN: 911210
AB To our knowledge, these are the first cases of almost complete Netherton's syndrome in two adult brothers born of consanguineous parents. The aromatic **retinoid**, etretinate, although initially worsened the eczematous manifestations, proved to be capable of reducing the primary skin lesions in one patient.
AB . . . these are the first cases of almost complete Netherton's syndrome in two adult brothers born of consanguineous parents. The aromatic **retinoid**, etretinate, although initially worsened the eczematous manifestations, proved to be capable of reducing the primary skin lesions in one patient.
CT Medical Descriptors:
***atopy**
*bamboo hair
*ichthyosis linearis circumflexa
*netherton disease
*skin defect
amino acid urine level
case report
consanguinity
heredity
diagnosis
therapy
human
*etretinate

L12 ANSWER 14 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 82246362 EMBASE
DN 1982246362
TI [Netherton's syndrome - a casuistic contribution. Treatment with the aromatic **retinoid** RO-9359].
DAS NETHERTON-SYNDROM - EIN KASUISTISCHER BEITRAG. BEHANDLUNG MIT DEM AROMATISCHEN **RETINOID** RO-9359.
AU Albrecht-Nebe H.; Reinicke C.; Thormann Th.
CS Dermatol. Klin., Bereich Med., Humboldt-Univ., 1040 Berlin, Germany
SO Dermatologische Monatsschrift, (1982) Vol. 168, No. 8, pp. 523-530.
CODEN: DMONBP
CY Germany
DT Journal
FS 037 Drug Literature Index
013 Dermatology and Venereology
LA German
SL English
ED Entered STN: 911209
Last Updated on STN: 911209
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER
TI [Netherton's syndrome - a casuistic contribution. Treatment with the aromatic **retinoid** RO-9359].
DAS NETHERTON-SYNDROM - EIN KASUISTISCHER BEITRAG. BEHANDLUNG MIT DEM AROMATISCHEN **RETINOID** RO-9359.
CT Medical Descriptors:
***atopy**
*bamboo hair
*ichthyosis linearis circumflexa
*netherton disease
therapy
adolescent

10676089

case report
*arotinoid
*etretinate
*nystatin

receptor agonists, are discussed. Molecules from members of this family have profound differentiating, antiproliferative, but. . .

L9 ANSWER 8 OF 23 MEDLINE on STN
AN 2002411073 MEDLINE
DN PubMed ID: 12164939
TI A highly decreased binding of cyclic adenosine monophosphate to protein kinase A in erythrocyte membranes is specific for active psoriasis.
AU Schopf Rudolf E; Langendorf Yvonne; Benz Roman E; Farber Lothar; Benes Peter
CS Department of Dermatology, Johannes Gutenberg University, Langenbeckstrasse 1, 55101 Mainz, Germany.. schopf@hautklinik.klinik.uni-mainz.de
SO Journal of investigative dermatology, (2002 Jul) 119 (1) 160-5. Journal code: 0426720. ISSN: 0022-202X.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200209
ED Entered STN: 20020808
Last Updated on STN: 20020918
Entered Medline: 20020917
AB A cyclic adenosine monophosphate binding abnormality in psoriatic erythrocytes that could be corrected by **retinoid** treatment has been reported. It was tested whether this binding abnormality is specific for psoriasis and the effects of treatment were compared with etretinate, cyclosporine A, or anthralin on 2-(3)H-8-N(3)-cyclic adenosine monophosphate binding to the regulatory subunit of protein kinase A in erythrocyte membranes. One hundred and fifteen individuals were evaluated, including: (i) 34 healthy persons; (ii) 15 patients with nonatopic inflammatory skin diseases (**eczema**, erythroderma, tinea, Grover's disease, erysipelas, urticaria); (iii) eight with other dermatoses mediated by immune mechanisms (systemic lupus erythematosus, lichen planus, necrotizing vasculitis, erythema nodosum, systemic sclerosis); (iv) 14 with generalized atopic dermatitis; and (v) 44 with psoriasis vulgaris clinically assessed by Psoriasis Area and Severity Index. In psoriasis, the course of the binding of 2-(3)H-8-N(3)-cyclic adenosine monophosphate to erythrocytes was measured in nine patients during a 10 wk treatment with etretinate, in 21 patients during a 10 wk treatment with cyclosporine A, and one patient under topical treatment with anthralin for 4 wk. We found the following femtomolar binding per mg protein: (i) healthy persons (1064 +/- 124, mean +/- SD); (ii) nonatopic inflammatory skin diseases (995 +/- 103); (iii) immune dermatoses (961 +/- 92); (iv) atopic dermatitis (960 +/- 110); and (v) psoriasis (645 +/- 159; $p < 0.0001$ compared with nonpsoriatics, Mann-Whitney U test). Treatment of psoriasis with etretinate, cyclosporine A, or anthralin normalized the binding of cyclic adenosine monophosphate, which was inversely correlated to the Psoriasis Area and Severity Index score. It was concluded that the decreased binding of cyclic adenosine monophosphate to protein kinase A in erythrocytes is specific for psoriasis and normalizes after successful treatment.
AB A cyclic adenosine monophosphate binding abnormality in psoriatic erythrocytes that could be corrected by **retinoid** treatment has been reported. It was tested whether this binding abnormality is specific for psoriasis and the effects of treatment. . . One hundred and fifteen individuals were evaluated, including: (i) 34 healthy persons; (ii) 15

patients with nonatopic inflammatory skin diseases (**eczema**, erythroderma, tinea, Grover's disease, erysipelas, urticaria); (iii) eight with other dermatoses mediated by immune mechanisms (systemic lupus erythematosus, lichen planus,. . .

CT . . .

therapeutic use

Middle Aged

Protein Binding: DE, drug effects

Protein Binding: PH, physiology

Psoriasis: DT, drug therapy

*Psoriasis: ME, metabolism

Retinoids: TU, therapeutic use

Severity of Illness Index

CN 0 (Affinity Labels); 0 (Anti-Inflammatory Agents); 0 (Azides); 0 (Dermatologic Agents); 0 (Keratolytic Agents); 0 (**Retinoids**); EC 2.7.1.37 (Cyclic AMP-Dependent Protein Kinases)